(d) applying an iterative process whereby various molecular structures are applied to said model to identify agonists or antagonists to said modified RTK polypeptide.

18. The method of claim 17 wherein said truncated kinase insert domain comprises a deletion of 50 residues from the KID.

19. The method of claim 17 wherein said truncated kinase insert domain comprises a deletion of 60 residues from the KID.

20. The method of claim 17 wherein said truncated kinase insert domain comprises a deletion of the highly charged residues from the KID.

21. The method of claim 17 wherein said truncated kinase domain linking said α helix D to said α helix E is of a sufficient length so as to allow said helices to maintain appropriate conformation associated with competent kinase structure.

22. The method of claim 17 wherein said RTK polypeptide is a member of the PDGFR family.

23. The method of claim 22 wherein said PDGFR member is selected from the group consisting of VEGFR-1, VEFGR-2, PDGFR-α, PDGFR-β, stem cell growth factor receptor (c-kit), and colony stimulating factor-1 receptor (CSF-1R/c-fms).

24. The method of claim 22 wherein said RTK polypeptide is selected from the group consisting of insulin receptor (IRK), fibroblast growth factor receptor-1 (FGFR-1), and VEGFR-2.

25. The method of claim 17 wherein said RTK polypeptide is VEGFR-2.

26. The method of claim 17 wherein said modified RTK polypeptide comprises VEGFR2Δ50 polypeptide of SEQ ID NO: 5.